Transient Q waves followed by left anterior fascicular block during exercise

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SUMMARY A 45 year old white man developed transient abnormal Q waves and ST segment elevation preceding left anterior fascicular block during exercise stress testing. The simultaneous disappearance of Q waves and fascicular block suggested that the abnormal Q waves were determined by an early septal conduction defect.

Transient abnormal Q waves have been described during selective cardiac surgery, hyperkalaemia, shock, severe metabolic stress, and acute coronary insufficiency. I Isolated reports exist about Q waves occurring during stress testing. We report a patient with two vessel coronary artery disease in whom exercise stress testing induced transient abnormal Q waves, ST segment elevation, and left anterior fascicular block.

Case report

A 45 year old white man was admitted to our ergometric laboratory for evaluation because of recurrent chest pain. For two years he had had infrequent episodes of a retrosternal burning sensation that radiated to the left arm and lasted for 10 to 15 minutes. He smoked 30 cigarettes a day.

On examination the resting heart rate was 67 beats/min and the resting blood pressure 120/80 mm Hg. The resting electrocardiogram showed (Fig. 1) sinus rhythm, QRS axis, +70°; QRS width, 80 ms; and RS pattern from V1 to V3 with T wave inverted in V1 and diphasic in V2.

Bicycle stress testing was performed with a work-load that was increased by 30 W at three minute intervals. After five minutes of exercise (60 W), at a heart rate of 130 beats/min and blood pressure of 185/105 mm Hg, he developed progressive chest pain; at that time a QS pattern appeared in V1-V2 and the R voltage was reduced in V3 simultaneously with ST segment elevation from V1 to V4. The QRS axis shifted to +30°, and the QRS width increased to 90 ms. The

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exercise test was stopped. At the fourth minute of the recovery period at a heart rate of 82 beats/min a further ST segment elevation in V2-V5, an increase in ORS width to 110 ms, and a shifting of QRS axis to -40° were noted. After 10 minutes rest at a heart rate of 70 beats/min the R waves in V1-V2 reappeared and the ORS axis and width returned to the pre-exercise values. Cardiac enzyme activities immediately and at three and six hours after exercise testing were normal (creatinine phosphokinase = 75 U/l, 85 U/l, and 72U/l, respectively). Cross sectional echocardiography, performed in the supine position and the left lateral decubitus using the apical approach at the time of interruption of the exercise testing, did not show any segmental wall motion abnormality at qualitative real time inspection. At the sixth minute of the recovery period, when the QRS axis was at -40°, apical echocardiography was repeated and did not show any modification (Fig. 2).

Coronary angiography performed four days later showed severe obstruction of the proximal left anterior descending and circumflex coronary arteries. At the time of bypass graft operation, performed three weeks later, no areas of hypokinesis were noted. Postoperatively he did well and the 12 lead electrocardiogram was normal.

Discussion

During stress testing, our patient developed transient Q waves in V1-V2 with ST segment elevation and subsequent left anterior fascicular block. No evidence confirming acute myocardial infarction was obtained.

In patients without previous myocardial infarction ST segment elevation during stress testing is considered to be indicative of Prinzmetal's angina, due to

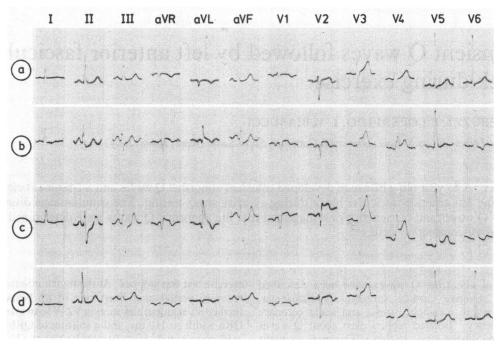


Fig. 1 (a) Resting electrocardiogram RS pattern from V1 to V3; QRS axis, $+70^{\circ}$. (b) During stress testing (fifth minute): QS pattern in V1 to V2; ST segment elevation from V1 to V4; QRS axis, $+30^{\circ}$. (c) Recovery (fourth minute): further ST segment elevation; left anterior fascicular block (QRS axis, -40°). (d) Recovery (tenth minute): reappearance of R waves in V1 to V2; disappearance of left anterior fascicular block.

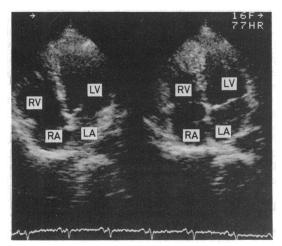


Fig. 2 Cross sectional echocardiogram in apical four chamber projection (left, diastole; right, systole), recorded at the sixth minute of the recovery period after exercise testing. No regional abnormalities in left ventricular wall motion are observed. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

severe proximal left anterior descending coronary artery obstruction, which was subsequently shown in our patient by coronary arteriography.³

Transient Q waves without acute myocardial infarction have been observed during coronary insufficiency, 4.5 Prinzmetal's angina, 3 and exercise electrocardiography. 2 According to the different authors, they may be explained either by electrical inertness of myocardial cells due to ischaemic alteration of the cell membrane 6.7 or by localised conduction disturbance. 8.9 In our patient an intraventricular conduction defect developed gradually after the appearance of Q waves; both Q waves and left anterior fascicular block disappeared simultaneously.

Transitory intraventricular conduction defects appearing during exercise testing may be rate related and not strictly dependent on the actual presence of acute myocardial ischaemia. ¹⁰ In our patient the QRS axis leftward shift developed at the peak of exercise, when the heart rate was fastest. The intraventricular conduction defect, however, did not disappear but increased after stopping the test and the lowering of the heart rate; therefore a rated related intraventricu-

lar conduction defect would be unlikely.

Regional wall motion abnormalities may be detected by cross sectional echocardiography or gated blood pool scanning in the course of acute myocardial infarction or transitory acute myocardial ischaemia.¹¹ Accurate phase analysis of wall motion by both methods may provide information on the modification of the excitability sequence or the presence of conduction abnormalities during myocardial ischaemia.¹²

In our patient only qualitative inspection of the apical echocardiogram was obtained, and it did not show any appreciable abnormality of wall motion movement or synchronism.

Even though definite proof of the nature of the phenomenon here described is lacking, in our opinion the simultaneous disappearance of Q waves and left fascicular block suggests that the abnormal Q waves were due to an early conduction defect localised in the septal area (septal fascicular block), which extended eventually to left anterior fascicular subdivisions. 13

The absence of any transitory wall motion abnormality at cross sectional echocardiography indicates electrophysiological rather than mechanical damage. Cellular inertness as the cause of the electrocardiographic sequence in our patient could not, however, be definitely excluded.

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